(FILE 'HOME' ENTERED AT 18:56:41 ON 04 DEC 2002)

FILE 'MEDLINE, BIOSIS, CANCERLIT, LIFESCI, BIOTECHDS, CAPLUS' ENTERED AT 18:57:11 ON 04 DEC 2002

L1 32607 S LIF OR ((LEUKEMIA OR LEUKAEMIA)(W)INHIBIT?(W)FACTOR#)
L2 25279 S (DIFFERENTI?)(5A)(LEUKEMI# OR LEUKAEMI# OR ERYTHROLEUKEMI# OR
L3 941 S L1 AND L2
L4 111 S L3 AND GLYCOPROTEIN
L5 48 DUP REM L4 (63 DUPLICATES REMOVED)
L6 340 S L1 AND AGGREGAT?
L7 79 S L1(5A)AGGREGAT?
L8 295 S L6 AND PY<1999
L9 5824 S D(A)FACTOR
L10 85 S L9 AND AGGREGAT?
L11 1 S L6 AND (AMID? OR DEAMID?)
L12 0 S L10 AND (AMID? OR DEAMID?)
L13 117 S L1(5A)(STABIL? OR (HALF(W)LIFE))
L14 17 S L9(5A)(STABIL? OR (HALF(W)LIFE))
L15 134 S L13 OR L14
L16 105 S L15 AND PY<1999
L17 93 DUP REM L16 (12 DUPLICATES REMOVED)
L18 3 S L17 AND (LEUKEMI# OR ERYTHROLEUKEMI# OR LEUKAEMI# OR ERYTHRO

L18 ANSWER 2 OF 3 MEDLINE
ACCESSION NUMBER: 81209972 MEDLINE
DOCUMENT NUMBER: 81209972 PubMed ID: 6972252
TITLE: Effect of tunicamycin on production by mouse fibroblast L929 cells of the factor-stimulating differentiation of mouse myeloid **leukemic** cells and the colony-stimulating factor.
AUTHOR: Yamamoto Y; Tomida M; Hozumi M; Ayusawa D; Seno T; Tamura G
SOURCE: CANCER RESEARCH, **(1981 Jun)** 41 (6) 2534-9.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198108
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19980206
Entered Medline: 19810827

AB Mouse myeloid **leukemic** M1 cells can be induced to differentiate into macrophages and granulocytes in vitro by a factor(s) stimulating differentiation of the cells (D-factor), which is suggested to be a glycoprotein. On the other hand, growth and differentiation of normal precursor cells of macrophages and granulocytes can be stimulated by a glycoprotein termed colony-stimulating factor (CSF). Mouse fibroblast L929 cells were found to produce both the D-factor and CSF. The properties of the D-factor and CSF and the roles of carbohydrates in the molecules of these factors were examined using tunicamycin, a specific inhibitor of asparaginase-linked glycosylation. Although both the D-factor and CSF were produced by L-cells in usual medium containing fetal calf serum, production of D-factor, but not CSF, was reduced by omission of serum from the medium. The activity of the D-factor was slightly decreased by treating the L-cells with tunicamycin (0.5 microgram/ml) in the presence of 2% fetal calf serum, without any decrease in CSF activity. Conditioned medium of L-cells incubated with or without tunicamycin was fractionated by gel filtration on a Sephadex G-200 column. Normal D-factor appeared as a single peak with an apparent molecular weight of 67,000. D-factor produced in the presence of tunicamycin had an apparent molecular weight of 25,000. On the other hand, most of the CSF was eluted in the void volume, even when it was produced in the presence of tunicamycin. The D-factor produced in the presence of tunicamycin was more sensitive than normal D-factor was to trypsin or heat treatment at 70 degrees. The CSF produced in the presence of tunicamycin was resistant to these treatments.

These results indicate that the D-factor is distinct from CSF. Furthermore, the results suggest that the D-factor produced by L-cells is also a glycoprotein and that, although carbohydrate is not essential for production or activity of the **D-factor**, it contributes to **stabilizing** the protein portion of **D-factor**.

L5 ANSWER 48 OF 48 MEDLINE DUPLICATE 24
ACCESSION NUMBER: 89048312 MEDLINE
DOCUMENT NUMBER: 89048312 PubMed ID: 3142300
TITLE: Purification of a murine **leukemia inhibitory factor** from Krebs ascites cells.
AUTHOR: Hilton D J; Nicola N A; Metcalf D
CORPORATE SOURCE: Walter and Eliza Hall Institute of Medical Research, Royal Melbourne Hospital Victoria, Australia.
SOURCE: ANALYTICAL BIOCHEMISTRY, (1988 Sep) 173 (2) 359-67. Journal code: 0370535. ISSN: 0003-2697.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198812
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19970203
Entered Medline: 19881214

AB A factor capable of inducing terminal **differentiation** in the murine myeloid **leukemia** cell line M1 has been purified to apparent homogeneity from the medium conditioned by Krebs II ascites tumor cells. The factor, termed **leukemia inhibitory factor** (**LIF**) is a single chain **glycoprotein** of apparent Mr 58,000 which induces differentiation and inhibits proliferation of the M1 cell line but not the WEHI-3B D+ murine myeloid leukemic cell line and has no detectable proliferative activity on normal myeloid progenitor cells. It was purified using four successive high-efficiency purification steps--anion-exchange chromatography on DEAE-Sepharose; cation-exchange chromatography on CM-Sepharose; affinity chromatography on lentil lectin-Sepharose; and reverse-phase high-performance liquid chromatography on a phenyl-silica matrix--to a specific biological activity of approximately 1.25 X 10(8) units/mg with an overall purification of 12,000-fold and a yield of 73% for the activity failing to bind to DEAE-Sepharose. Sufficient quantities of the factor (12 micrograms, 200 pmol) have been purified to allow structural and functional analysis of the molecule and comparison with other know differentiation inducers.

L5 ANSWER 46 OF 48 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1988:376457 BIOSIS
DOCUMENT NUMBER: BA86:60367
TITLE: RESOLUTION AND PURIFICATION OF THREE DISTINCT FACTORS PRODUCED BY KREBS ASCITES CELLS WHICH HAVE **DIFFERENTIATION**-INDUCING ACTIVITY ON MURINE MYELOID **LEUKEMIC** CELL LINES.
AUTHOR(S): HILTON D J; NICOLA N A; GOUGH N M; METCALF D
CORPORATE SOURCE: WALTER AND ELIZA HALL INST. MED. RES., P.O. ROYAL MELBOURNE HOSP. 3050, VICTORIA, AUST.
SOURCE: J BIOL CHEM, (1988) 263 (19), 9238-9243.
CODEN: JBCHA3. ISSN: 0021-9258.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB The use of different myeloid leukemic cell lines (WEHI-3B D+ and M1) and different sources of factors has led to discrepancies concerning the identity of factors capable of inducing **differentiation** in **leukemic** cells. We have biochemically fractionated medium conditioned by one such source (Krebs II ascites cells) and assayed fractions for their bone marrow colony-stimulating activity as well as their differentiation-inducing activity for WEHI-3B D+ and M1 cells. This resulted in the resolution of four distinct molecular species with differentiation-inducing activity. One activity was purified to homogeneity and shown by a variety of biochemical, biological, and receptor-binding criteria to be authentic granulocyte colony-stimulating factor (G-CSF). A second activity was identified as granulocyte-macrophage colony-stimulating factor (GM-CSF). Two other activities termed **LIF**-A and **LIF**-B (**leukemia inhibitory factor**) were shown to probably be different glycosylation variants of the same protein and one of these (**LIF**-A) was purified 12,000-fold to homogeneity. G-CSF induced differentiation in both WEHI-3B D+ and at higher concentrations M1 cells while GM-CSF weakly induced differentiation in WEHI-3B D+ cells. **LIF**-A had no colony-stimulating activity and induced differentiation in and inhibited the proliferation of only M1 cells. Each factor bound to a unique cell surface receptor with no evidence of direct cross-reactivity.

L5 ANSWER 44 OF 48 MEDLINE DUPLICATE 21
ACCESSION NUMBER: 90200634 MEDLINE
DOCUMENT NUMBER: 90200634 PubMed ID: 2180647
TITLE: Protein factors that regulate the growth and **differentiation** of mouse myeloid **leukaemia** cells.
AUTHOR: Hozumi M; Tomida M; Yamamoto-Yamaguchi Y; Kasukabe T; Okabe-Kado J; Honma Y; Hayashi M
CORPORATE SOURCE: Department of Chemotherapy, Saitama Cancer Center Research Institute, Japan.
SOURCE: CIBA FOUNDATION SYMPOSIUM, (1990) 148 25-33; discussion 33-42. Ref: 28
Journal code: 0356636. ISSN: 0300-5208.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199005
ENTRY DATE: Entered STN: 19900601
Last Updated on STN: 19900601
Entered Medline: 19900508

AB We have purified and characterized several protein factors that regulate the growth and **differentiation** of mouse myeloid **leukaemia** M1 cells. The **differentiation** factor (D-factor) from conditioned medium (CM) of Ehrlich ascites tumour cells is a **glycoprotein** of Mr 40,000-50,000. Its amino acid sequence was found to be almost identical to that of **leukaemia** **inhibitory factor** (**LIF**) from Krebs II ascites cells. The differentiation inhibitory factor (I-factor) from the CM of variant M1 cell clones which were resistant to several differentiation inducers is a basic protein of apparent Mr 68,000. The growth inhibitory factor (GI-factor) that specifically inhibits the partially **differentiated** and still growing monocytic **leukaemia** M1 cells was isolated from the CM of a clone of M1 cells resistant to the differentiation inducers. This GI-factor is a basic protein with an Mr of 25,000. Regulation by these protein factors together with other known cytokines of growth and differentiation of M1 cells is reported.

L5 ANSWER 42 OF 48 MEDLINE
ACCESSION NUMBER: 90253815 MEDLINE
DOCUMENT NUMBER: 90253815 PubMed ID: 2111155
TITLE: Localization of the murine **leukemia inhibitory factor** gene near the centromere on chromosome 11.
AUTHOR: Kola I; Davey A; Gough N M
CORPORATE SOURCE: Centre for Early Human Development, Monash Medical Center, Clayton, Victoria, Australia.
CONTRACT NUMBER: CA-22556 (NCI)
SOURCE: GROWTH FACTORS, (1990) 2 (2-3) 235-40.
Journal code: 9000468. ISSN: 0897-7194.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199006
ENTRY DATE: Entered STN: 19900720
Last Updated on STN: 19900720
Entered Medline: 19900625

AB **Leukemia inhibitory factor** (**LIF**) is a **glycoprotein** with divergent activities: It induces the **differentiation** of certain myeloid **leukemic** cells, inhibits the **differentiation** of embryonic stem cells, and promotes bone remodelling in vivo and in vitro. The murine **LIF** gene has been assigned to the proximal region of chromosome 11 at sub-bands A1-A2, by analysis of a panel of mouse x Chinese hamster somatic cell hybrids and by in situ hybridization. Interestingly, the proximal portion of chromosome 11 has been shown, by virtue of its parental origin effects, to contain gene(s) involved in fetal growth. It is also interesting that there is a preponderance of chromosome 11 abnormalities in embryonal carcinoma cells. The localization of the murine **LIF** gene confirms the homology of a portion of murine chromosome 11 with human chromosome 22q, the site of the human **LIF** gene.

L5 ANSWER 41 OF 48 LIFESCI COPYRIGHT 2002 CSA
ACCESSION NUMBER: 93:100886 LIFESCI
TITLE: Developmentally programmed induction of differentiation inhibiting activity and the control of stem cell populations.
AUTHOR: Rathjen, P.D.; Nichols, J.; Toth, S.; Edwards, D.R.; Heath, J.K.; Smith, A.G.
CORPORATE SOURCE: CRC Growth Factors Group, Dep. Biochem., Univ. Oxford, Oxford OX1 3QU, UK
SOURCE: GENES DEV., (1990) vol. 4, no. 126, pp. 2308-2318.
DOCUMENT TYPE: Journal
FILE SEGMENT: F
LANGUAGE: English
SUMMARY LANGUAGE: English

AB **Differentiation** inhibiting activity/**leukemia inhibitory factor** (DIA/**LIF**) is a **glycoprotein** that controls differentiation of pluripotential stem cells. Alternative transcription generates both diffusible and matrix-associated forms of DIA/**LIF**. Transcriptional analysis using a sensitive ribonuclease protection assay revealed that the two messages are expressed independently, consistent with the proposition that the two forms of DIA/**LIF** have distinct biological roles. DIA/**LIF** expression was found to be activated early during differentiation of embryonic stem (ES) cells, providing a mechanism for feedback regulation of stem cell renewal. Expression of DIA/**LIF** by mesenchymal cells was shown to be controlled in a paracrine manner by polypeptide regulatory factors.

L5 ANSWER 40 OF 48 MEDLINE DUPLICATE 19
ACCESSION NUMBER: 91122633 MEDLINE
DOCUMENT NUMBER: 91122633 PubMed ID: 1703981
TITLE: Developmentally programmed induction of differentiation inhibiting activity and the control of stem cell populations.
AUTHOR: Rathjen P D; Nichols J; Toth S; Edwards D R; Heath J K; Smith A G
CORPORATE SOURCE: Department of Biochemistry, University of Oxford, UK.
SOURCE: GENES AND DEVELOPMENT, (1990 Dec) 4 (12B) 2308-18. Journal code: 8711660. ISSN: 0890-9369.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199103
ENTRY DATE: Entered STN: 19910405
Last Updated on STN: 19960129
Entered Medline: 19910314

AB **Differentiation** inhibiting activity/**leukemia inhibitory factor** (DIA/**LIF**) is a **glycoprotein** that controls differentiation of pluripotential stem cells. Alternative transcription generates both diffusible and matrix-associated forms of DIA/**LIF**. Transcriptional analysis using a sensitive ribonuclease protection assay revealed that the two messages are expressed independently, consistent with the proposition that the two forms of DIA/**LIF** have distinct biological roles. DIA/**LIF** expression was found to be activated early during differentiation of embryonic stem (ES) cells, providing a mechanism for feedback regulation of stem cell renewal. Expression of DIA/**LIF** by mesenchymal cells was shown to be controlled in a paracrine manner by polypeptide regulatory factors. Specific expression of the two forms of DIA/**LIF** was also demonstrated in the egg cylinder-stage mouse embryo. The combination of cell type-specific and signal-specific regulation enables very precise control over DIA/**LIF** expression and may represent an important component of the regulatory networks that govern stem cell proliferation and differentiation during mammalian development.

L5 ANSWER 35 OF 48 MEDLINE DUPLICATE 15
ACCESSION NUMBER: 92020908 MEDLINE
DOCUMENT NUMBER: 92020908 PubMed ID: 1717982
TITLE: Oncostatin M is a member of a cytokine family that includes **leukemia-inhibitory factor,** granulocyte colony-stimulating factor, and interleukin 6.
AUTHOR: Rose T M; Bruce A G
CORPORATE SOURCE: Fred Hutchinson Cancer Research Center, Seattle, WA 98104.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1991 Oct 1) 88 (19) 8641-5. Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199110
ENTRY DATE: Entered STN: 19920124
Last Updated on STN: 19960129
Entered Medline: 19911031

AB Oncostatin M (OSM), a **glycoprotein** of Mr approximately 28,000 produced by activated monocyte and T-lymphocyte cell lines, was previously identified by its ability to inhibit the growth of cells from melanoma and other solid tumors. We have detected significant similarities in the primary amino acid sequences and predicted secondary structures of OSM, **leukemia-inhibitory factor (LIF),** granulocyte colony-stimulating factor (G-CSF), and interleukin 6 (IL-6). Analysis of the genes encoding these proteins revealed a shared exon organization, suggesting evolutionary descent from a common ancestral gene. Using a panel of DNAs from somatic cell hybrids, we have shown that OSM, like **LIF,** is located on human chromosome 22. We have also demonstrated that OSM has the ability to inhibit the proliferation of murine M1 myeloid **leukemic** cells and can induce their **differentiation** into macrophage-like cells, a function shared by **LIF,** G-CSF, and IL-6. We propose that OSM, **LIF,** G-CSF, and IL-6 are structurally related members of a cytokine family that have in common the ability to modulate differentiation of a variety of cell types.

L5 ANSWER 29 OF 48 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:75172 CAPLUS
DOCUMENT NUMBER: 120:75172
TITLE: Soluble interleukin-6 receptor triggers osteoclast formation by interleukin 6
AUTHOR(S): Tamura, Tatsuya; Udagawa, Nobuyuki; Takahashi, Naoyuki; Miyaura, Chisato; Tanaka, Sakae; Yamada, Yoshiki; Koishihara, Yasuo; Ohsugi, Yoshiyuki; Kumaki, Kenji; et al.
CORPORATE SOURCE: Sch. Dent., Showa Univ., Tokyo, 142, Japan
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (1993), 90(24), 11924-8
CODEN: PNASA6; ISSN: 0027-8424
DOCUMENT TYPE: Journal
LANGUAGE: English

AB It has been reported that sol. interleukin (IL)-6 receptor (sIL-6R) is detected in the serum of healthy individuals and its level is increased in patients with multiple myeloma and human immunodeficiency virus infection.

Although several reports have suggested that sIL-6R potentiates IL-6 action, is physiol. role remains unclear. In this study, the authors examd. the role of sIL-6R on osteoclast formation by IL-6, using a coculture of mouse osteoblasts and bone marrow cells. Neither recombinant mouse IL-6 (mIL-6) nor mouse sIL-6R (smIL-6R) induced osteoclast-like multinucleated cell (MNC) formation when they were added sep. In contrast, simultaneous treatment with mIL-6 and smIL-6R strikingly induced MNC formation. These MNCs satisfied major criteria of authentic osteoclasts, such as tartrate-resistant acid phosphatase (TRAP) activity, calcitonin receptors, and pit formation on dentin slices. The MNC formation induced by mIL-6 and smIL-6R was dose-dependently inhibited by adding monoclonal anti-mouse IL-6R antibody (MR16-1). It is likely that osteoblasts and osteoclast progenitors are capable of transducing a signal from a complex of IL-6 and sIL-6R through gp130, even though they may have no or a very small no. of IL-6Rs. Factors such as IL-11, oncostatin M, and **leukemia inhibitory factor**, which are known to exert their functions through gp130 (the signal-transducing chain of IL-6R), also induced MNC formation in the authors' coculture system. These results suggest that increased circulating or locally produced sIL-6R induces osteoclast formation in the presence of IL-6 mediated by a mechanism involving gp130. This may play an important physiol. or pathol. role in conditions assocd. with increased osteoclastic bone resorption.

L5 ANSWER 21 OF 48 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:292859 CAPLUS
DOCUMENT NUMBER: 126:329104
TITLE: Regulation of oligodendrocyte cell survival and **differentiation** by ciliary neurotrophic factor, **leukemia inhibitory factor**, oncostatin M, and interleukin-6
AUTHOR(S): Vos, J. P.; Gard, A. L.; Pfeiffer, S. E.
CORPORATE SOURCE: Department of Microbiology, University of Connecticut Medical School, Farmington, CT, USA
SOURCE: Perspectives on Developmental Neurobiology (1996), 4(1), 39-52, 1 Plate
CODEN: PDENED; ISSN: 1064-0517
PUBLISHER: Gordon & Breach
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with 133 refs. The regulation and maintenance of developmental lineages by trophic factors, both cell-mediated and sol., is a key aspect of cellular differentiation in the nervous system. In this review we focus on oligodendrocytes and their progenitors and how differentiation and survival are regulated by four neuropoietic cytokines: ciliary neurotrophic factor, **leukemia inhibitory factor**, oncostatin M, and interleukin-6 (IL-6). We discuss how these cytokines act as "broad spectrum" factors. I.e., how, even within a specific cell lineage, a given cytokine may have different effects on the target cells at various stages of differentiation.

L5 ANSWER 22 OF 48 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:556801 CAPLUS
DOCUMENT NUMBER: 122:288671
TITLE: Cardiotrophin-1. Biological activities and binding to the **leukemia inhibitory factor** receptor/gp130 signaling complex
AUTHOR(S): Pennica, Diane; Shaw, Kenneth J.; Swanson, Todd A.; Moore, Mark W.; Shelton, David L.; Zioncheck, Kimberly A.; Rosenthal, Arnon; Taga, Tetsuya; Paoni, Nicholas F.; Wood, William I.
CORPORATE SOURCE: Dep. Molecular Biol., Cell Genetics, Neurobiol., Immunology, South San Francisco, CA, 94080, USA
SOURCE: Journal of Biological Chemistry (1995), 270(18), 10915-22
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Cardiotrophin-1 (CT-1) is a newly isolated cytokine that was identified based on its ability to induce cardiac myocyte hypertrophy. It is a member of the family of cytokines that includes interleukins-6 and -11, **leukemia inhibitor factor** (**LIF**), ciliary neurotrophic factor, and oncostatin M. These cytokines induce a pleiotropic set of growth and differentiation activities via receptors that use a common signaling subunit, gp130. In this work we det. the activity of CT-1 in six in vitro biol. assays and examine the compn. of its cell surface receptor. We find that CT-1 is inactive in stimulating the growth of the hybridoma cell line, B9 and inhibits the growth of the mouse myeloid leukemia cell line, M1. CT-1 induces a phenotypic switch in rat sympathetic neurons and promotes the survival of rat dopaminergic and chick ciliary neurons. CT-1 also inhibits the differentiation of mouse embryonic stem cells. CT-1 and **LIF** cross-compete for binding to M1 cells, Kd [CT-1] .apprx. 0.7 nM, and this binding is inhibited by an anti-gp130 monoclonal antibody. Both ligands can be specifically cross-linked to a protein on M1 cells with the mobility of the **LIF** receptor (.apprx.200 kDa). In addn., CT-1 binds directly to a purified, sol. form of the **LIF** receptor in soln. (Kd .apprx. 2 nM). These data show that CT-1 has a wide range of hematopoietic, neuronal, and developmental activities and that it can act via the **LIF** receptor and the gp130 signaling subunit.

L5 ANSWER 7 OF 48 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 2000078517 MEDLINE
DOCUMENT NUMBER: 20078517 PubMed ID: 10613356
TITLE: Induction of gp130 and **LIF** by **differentiation** inducers in human myeloid **leukemia** K562 cells.
AUTHOR: Xie P; Chan F S; Ip N Y; Leung M F
CORPORATE SOURCE: Department of Biology, Biotechnology Research Institute, The Hong Kong University of Science and Technology, People's Republic of China.
SOURCE: LEUKEMIA RESEARCH, (1999 Dec) 23 (12) 1113-9. Journal code: 7706787. ISSN: 0145-2126.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200001
ENTRY DATE: Entered STN: 20000124
Last Updated on STN: 20000124
Entered Medline: 20000110

AB It has been previously shown that phorbol 12-myristate 13-acetate (PMA), a potent differentiation inducer, induced the expression of both interleukin-6 (IL-6) and IL-6 receptor alpha component (IL-6Ralpha) in K562 leukemia cells. In the present study, we examined the ability of several differentiation inducers to regulate the expression of the signal-transducing receptor component for IL-6, gp130, and cytokine **leukemia inhibitory factor** (**LIF**) in K562 cells. We found that the expression of gp130 was dramatically induced at both the mRNA and protein levels by the two megakaryocytic inducers sodium butyrate (NaBut) and PMA. In contrast, the mRNA expression of **LIF** was induced by the two erythroid inducers 1-beta-D-arabinofuranosyl cytosine (Ara-C) and hemin. Furthermore, activation of the PMA-induced gp130 receptor by exogenous IL-6 potentiated the differentiating effects of PMA. Our findings suggest that IL-6/gp130 signaling may be involved in the regulation of the megakaryocytic differentiation of K562 cells.